PYORRHŒA ALVEOLARIS.

FITZGERALD.

PYORRHŒA ALVEOLARIS

AND ITS RELATIONS TO GENERAL MEDICINE.

BY

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OPINIONS OF THE PRESS.

"In this small volume the author gives a brief yet lucid account of the condition termed 'Pyorrhœa Alveolaris.' It is extremely useful in bringing together in a concise form the *principal facts which are at present known. The remarks on treatment are good.*"—*Lancet.*

"The author describes this well-known though frequently neglected condition of inflammation of the gums, which gives rise, in all probability, to many of the cases of chronic disease of the mucous membranes of the lips and tongue, the causation of which is so often obscure."—*British Journal of Dermatology.*

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"The present booklet is a reprint of a series of articles which appeared in the *Clinical Journal* during March and April, 1899. The subject of pyorrhœa alveolaris, or Riggs' disease, is one which has been almost entirely confined to the dental profession. As we have had occasion to point out on numerous occasions, *it is a trouble with which every physician should familiarise himself, and in whose study every dentist should perfect himself.* The disease is not a mere local process, but most often an expression of the presence of some dyscrasia. In the little monograph before us the author sets forth these points very clearly, *and it cannot fail of being of the highest value to both the medical and the dental professions.* We are very much pleased with it, and can recommend it heartily."—*Medical and Surgical Journal of St. Louis.*

PREFACE TO THE SECOND EDITION.

On account of the rapid exhaustion of the first edition of this work, such a short period has elapsed since its original publication that it has not been considered necessary to make either additions or alterations.

This work does not profess to be an exhaustive treatise upon "Pyorrhœa Alveolaris," but rather a summary of present knowledge and methods of treatment.

It is also intended to point out the bearings of this disease upon Medical practice, and to indicate to the practitioner the treatment of the condition.

It is a reprint of a series of articles which appeared in the 'Clinical Journal' during March and April, 1899.

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PYORRHŒA ALVEOLARIS AND ITS RELATIONS TO GENERAL MEDICINE.

Pyorrhœa alveolaris in its strictest sense is a term which may be applied to any diseased condition of the gums which is accompanied by the discharge of pus from them into the mouth. As a matter of fact its use is confined to certain well-defined affections of the tissues surrounding the teeth.

It was undoubtedly first described by Dr. J. M. Riggs, of Hartford, Conn., U.S.A., and must have been recognised by him as early as 1844, since in a paper read by him at a meeting of the Connecticut Valley Dental Association in June, 1869, he stated that he had treated this condition with success for over twenty-five years. It has been from that time

associated with his name, and called Riggs' disease. Subsequently other names have been proposed, such as expulsive gingivitis, alveolar periostitis, and phagedænic pericementitis. The term pyorrhœa alveolaris was proposed by Dr. Rehwinkle at a meeting of the American Dental Association in 1877, but it appears to have been previously used by Wedl in a paper published several years previously.

Pyorrhœa alveolaris is in many ways an unfortunate name, as it only describes one feature of the later stages of the affection. Nevertheless it is now so firmly established that we may usefully retain it as a convenient term. Before studying the pathology of pyorrhœa, it will perhaps be as well to briefly review the manner in which the teeth are fastened in their sockets.

The teeth, as we all know, are received into sockets in the alveolar processes of the maxillary bones. Between the teeth and the walls of the aforesaid sockets is interposed a substance called the peridental membrane. This is composed of fibrous elastic tissue, and in fact may be looked upon as a

suspensory ligament of the tooth, as it allows a limited amount of motion. Besides supporting and fixing the tooth in its position it furnishes nourishment to the bony surfaces between which it is placed —the inner wall of the alveolus, and the outer layer of cementum on the tooth. For this latter office it is well supplied with blood-vessels from two sources. The artery which enters the apex of the tooth gives off a branch which enters it below; whilst above, the gums send vessels to it over the margin of the socket of the tooth. At its upper margin the peridental membrane forms a firm attachment between the neck of the tooth and the periosteum of the alveolar wall, such attachment being from one eighth to three eighths of an inch from the free margin of the gums. The free margin of the gum is covered on its exposed surface by a very dense squamous epithelium which is able to withstand the abrading action of the food during the act of mastication. Under this is a layer of softer epithelial cells, which cover a series of papillary projections from the subjacent fibrous tissue. The epithelium which covers the inner

surface of the gum margin, *i. e.* that portion in contact with the neck of the tooth, consists of very soft round or polygonal cells, and is of considerable depth. Although not of glandular structure, it secretes a profusion of small rounded cells, which are to be found in abundance in the saliva, and are usually called mucous corpuscles. For this reason it has been termed the gingival organ. It is probable that this structure is not the sole source of the mucous cells in the saliva, but it will certainly account for many of them. This can easily be proved by passing the end of a thin instrument under the free margin of the healthy gum, and transferring the secretion thus obtained to a slide for microscopic examination. The knowledge of this organ and its secretion is of the greatest importance to the physician; as the accumulation of these cells mixed with organisms under the free margin of the gum forms soft cheesy masses that so nearly resemble pus, that they may be easily mistaken for it, and thus lead the observer to make an erroneous diagnosis of pyorrhœa.

The production of pyorrhœa depends upon two factors, a predisposing cause and a local irritation.

The predisposing cause may be tubercle, syphilis, scurvy, the exhaustion of acute infectious diseases, or any other source of malnutrition.

The exciting cause may be, and most usually is, a gingivitis produced in one of the ways to be presently described.

There is also a pyorrhœa of gouty origin, in which the local necrosis of the peridental membrane is caused by gouty disease of one of the blood-vessels in its substance.

Pyorrhœa is met with at any age, and it is quite erroneous to consider that we only see it after thirty-six years of age. Of course young people will not suffer from it in the degree that older ones do, because there has not been so long a time for the affection to have developed.

For many years the association between gout and pyorrhœa has been noticed, and a certain relation suspected. The chief advocate of this theory is

Professor Pierce, who in a paper published in the 'Dental Practitioner' in April, 1894, enunciated the theory that pyorrhœa was nearly always a local expression of a constitutional diathesis, and that the mechanism of its causation was some morbid constituent of the blood, which exuded and infiltrated the alveolo-cemental membrane, the effect of this being to set up a pericemental irritation commencing at or near the apical extremity of the root; and he considers it proved that this irritating material is of a gouty nature, from the fact that as the result of a series of chemical analyses made by him, the concretions which in these cases are found on tho roots near the apices, were found to consist largely of uric acid and urates.

As against his theory it has been argued by Burchard and others, that the deposit could not take place upon the root of the tooth except from the lining membrane of the alveolus, since this is in contact with the tooth and practically attached to it. Talbot also ('Dental Cosmos,' vol. xxxviii, p. 310) undertook a series of experiments, and only found

uric acid upon the deposits of very few teeth out of those which he examined. It is, however, almost certain that Pierce is right in the main, but that the uric acid deposits are the result and not the cause of the gouty inflammation. One has only to bear in mind that the alveolo-cemental membrane is a vascular fibrous tissue, and is the analogue of the ligamentous structures in joints, to understand how likely it is that gouty inflammation of it should take place. We know that over-use of a joint will often determine an attack of gout in it; and the pericemental membrane being a true suspensory ligament of the teeth, and allowing a really considerable amount of movement in them, may be affected in the same way as other joints—for the tooth in its socket may be properly regarded as a joint. Moreover, from defective position of the teeth, mal-occlusions and defects in the bite, the suspensory ligament is constantly undergoing considerable strain, and we may reasonably expect that in a gouty person there may be local inflammation set up. In these cases there will probably be first inflammatory thickening of the

vessels supplying the pericemental membrane of a gouty nature. This will be followed by thickening of their walls from swelling of the tunica media in the usual way observed in gouty inflammation. There will then ensue obliteration of its lumen, and death of the portion of tissue to which it is supplied, with a local molecular necrosis and the subsequent deposition of urates.

It appears more than probable that syphilis may account for a certain number of the cases of pyorrhœa which are met with in practice. One observer found that out of *twenty-three syphilitic patients, eleven had true pyorrhœa.* In these cases there are probably two factors at work, first the constitutional weakness, which is the direct result of the disease, and secondly the gingivitis, which it is almost impossible to avoid when administering mercurial preparations. Although the greatest care may be taken to prevent salivation, yet the soreness of the gums which is almost always produced, and which is generally accepted as the indication that the proper dose has been reached, will be quite sufficient to impair the integrity of the peri-

dental membrane and open the way to the invasion of pyogenic organisms.

Disease of the peridental membrane and subsequent pyorrhœa appears to arise in one of the following ways : as the result of a gingivitis, following death of the pulp of the tooth, or from gouty disease in the substance of the membrane itself.

All these lead up to the same end, but by slightly different paths. We will study them in rotation.

The Result of a Gingivitis.

We may conveniently discuss pyorrhœa commencing in this manner under several heads which form distinct stages in the progress of the affection.

(*a*) The predisposing cause.

(*b*) The local irritation which is the immediate cause of the gingivitis.

(*c*) The gingivitis itself.

(*d*) The streptococcus invasion, and

(*e*) The absorption of bony tissue.

(*a*) *The predisposing cause.*—As predisposing causes

of pyorrhœa we may have the causes which predispose to the antecedent gingivitis, or which may render the peridental membrane less resisting. Thus we may have an hereditary tendency to catarrhal affections or chronic nasal obstruction with its induced catarrhal condition of mouth and fauces.

On the other hand, pyorrhœa may well be predisposed to by any condition which will depress the general nutrition, since the circulation will fail first in the remotest capillaries, such as those in the peridental membrane. These tissues will be thus rendered less resistent. We may thus find as factors, lactation, hæmorrhages, syphilis, tubercle, and convalescence from acute specific fevers. It is also extremely probable that in nervous affections such as tabes and rheumatoid arthritis, which produce or are accompanied by trophic changes, there may be an increased tendency to degenerative processes in the peridental membrane.

It is obvious that in anæmia the resisting power of the peridental membrane must be greatly lessened from the defective circulation in the gums.

(*b*) *The local irritation.*—It appears to be quite certain that the gingival organs possess, in common with some other tissues of the body, the power of selecting and excreting poisonous substances from the blood. It is also well known that some of these cause hyperæmia or even inflammation in their passage. We have notable examples of this in the inflammation of the gums caused by mercury and iodide of potassium. Although this is really and essentially a gingivitis, yet it is usually spoken of as salivation, owing to the fact that the salivary glands are also affected. In its slighter forms, as met with in patients who are undergoing systematic treatment, it is a gingivitis pure and simple. The exciting cause of a gingivitis, then, may be a mercurial course.

Dentists are responsible for many cases of gingivitis. In some instances they directly inoculate the gums with disease germs by performing dental operations without first *sterilising* the *mouth*. They confine their attention to disinfecting the instruments, and forget that the mouth itself is full of disease germs waiting to take advantage of any laceration of

surface. In other cases, by means of improperly fitting crowns, fillings overhanging at the neck of the tooth, crude and badly finished amalgam stoppings, and badly constructed and ill-fitting plates, they keep up such a continual irritation of the edges of the gums that gingivitis soon occurs.

Gingivitis is very commonly induced by the irritation of lactic acid formed by the fermentation of food residues retained under the margins of the gums. This generally occurs in people who do not brush the teeth at night when retiring to bed. It is much more important to cleanse the teeth at bedtime than in the morning. It is in the long period during the night, when the food débris is undisturbed, that opportunity arises for fermentation to take place.

Gingivitis is also produced by tartar upon the teeth. Every one is aware that tartar is deposited upon the teeth, but very few understand why and how this comes to pass, and wonder why it should accumulate upon some teeth, and not on others. It is now pretty well established that calcareous deposit in any part of the body is effected by the agency of

an organic material such as mucin. Without the presence of such a substance the earthy salts cannot be thrown down from the animal fluids which hold them in solution in such a form as to produce an adherent deposit. The calcium salts are apparently held in solution in the saliva by means of carbonic acid gas, and are thrown down on coming in contact with the air. If any mucin is present to bind them, calculi will be formed, which will adhere to any suitable adjacent surface, especially if there is also present a local roughness to assist the adhesion. The mystery of the deposition of tartar in the mouth is thus easily explained, and, as will be seen, it is most frequent just where we should *a priori* expect to find it, viz. in the neighbourhood of the ducts of the glands, which pour mucin-containing secretions into the mouth.

One of the most frequent sites for the deposition of tartar is on the buccal surfaces of the back teeth in the upper jaw. A little reflection will remind us that this is opposite the spot where the parotid glands, by means of Steno's duct, pour their secretion

into the mouth. The parotid saliva differs from the secretion of the other glandular tissue in the mouth in containing a relatively small proportion of organic material, and this in the form of a globulin rather than a mucin. The consequence, as we should expect, is that the calculus formed by its aid differs in character from that deposited upon teeth in other situations, and is softer and more easily broken down.

But in order that tartar should be deposited in this situation another factor is essential, and that is that the parotid secretion should have an opportunity of remaining undisturbed in contact with the teeth on which the deposit is to take place. This happens when from defect of the lower molars the upper ones are practically not used in mastication. In such cases the saliva remains, and stagnates in the pocket formed by the reflection of the mucous membrane outside and above these teeth, and this region not as a rule being reached by the tooth-brush, every condition requisite for an abundant deposition of tartar is afforded.

The same combination of circumstances obtains to a great extent under the tongue, and as a consequence a plentiful deposit of tartar takes place on the lingual surfaces of the lower incisors and laterals. It is important to note that whilst in healthy mouths the tendency to the deposit of tartar is comparatively slight, yet the presence of even a slight degree of gingivitis will enormously increase the process. And this is easily understood when we reflect that such an inflammation of the gums will add a relatively large amount of proteid material to the saliva in these situations. The formation of calcareous deposits under the edges of the gums is thus accounted for. The effect of the gingivitis will be to slightly evert and separate the edges of the gums from the neck of the tooth, and in the cavity thus formed the saliva mixed with the albuminous discharge of the gingivitis will cause a deposit limited in extent of very hard and adherent tartar.

After tartar has once been deposited upon the neck of a tooth, a vicious circle is established which is very hard to break. When a slight deposit has once

taken place it becomes an irritant which will itself perpetuate the condition. It is a remarkable fact that all varieties of tartar appear to possess extremely irritating qualities which keep the border of the peridental membrane and the adjacent gum in a continual state of chronic inflammation, with the result that the deposit slowly and continuously increases. The effect of the irritation of the deposit of tartar is to cause a change of a progressive nature in the peridental membrane, which undergoes degeneration, as we shall presently show, with the formation of a cavity, or as it is technically termed a pocket.

The effect of the tartar in eroding the gum is, I think, due to two factors :

1. Abundance of lactic acid is generated in the food débris which accumulates in the space between the edge of the tartar and the gum. All starchy food contains lactic acid germs, as one can readily demonstrate in the following manner. Take some crumb of bread and mix with warm water and keep at the temperature of the body for a couple of hours or so. Express the liquid from it, and titrate it with a deci-

normal solution of sodium hydrate, using a few drops of an alcoholic solution of phenol-phthalein to mark the end of the reaction. The total acidity will by this means be found to be considerable. This can be demonstrated to be mainly due to lactic acid by the ordinary method with Uffelman's test. This lactic acid probably acts upon the gum, making it more vulnerable.

2. The absolute pressure of the tartar will erode the gums. The immediate effect of the pressure of the rough unyielding tartar upon the spongy inflamed gum is, by the irritation set up, to cause it to grow. The swollen gum then presses in its turn against the lower edge of the tartar until the result is that its vitality is destroyed, and a breach of surface takes place.

Besides the tartar deposited from the saliva upon the teeth, we have another variety which occurs under the margins of the gums. This has been termed serumic calculus. This name was given to it because it was supposed to be formed from the serum of the blood. It is more probable that in the case

of gums not affected with gingivitis, it is deposited from the secretion of the tubular epithelial glands of the peridental membrane. It may then set up a gingivitis, the immediate result of which will be the deposit of fresh nodules of tartar of a slightly different character derived from the secretions of the diseased surface. It is an unfortunate fact, tending to obscure an intelligent understanding of these concretions, that most writers on dental subjects apply the term serumic calculus indiscriminately to all nodules of tartar occurring upon the roots of teeth. This is obviously wrong, as deposits may arise in quite a different manner. An example may be found in the scales found on the roots of teeth where the disease has originated in gouty disease of a portion of the peridental membrane itself. Under these circumstances the deposit of tartar appears to arise in obedience to the general law which obtains in pathological processes in the body, that dead tissue appears to attract in some mysterious way the calcium salts of the blood. In some cases, probably in all, this is a wonderful effort on the part of nature either to repair the

damage or to shut the diseased area off from the rest of the body, and to confine the morbid process within the enclosed area. We have an example of this in the calcification which takes place under favourable circumstances in tubercular disease of the lung. But, unfortunately, it is impossible for nature to adjust the mechanism so that it can provide for all contingencies, and so we have the aimless deposit of lime salts in positions where it can not only be of no possible service, but even do positive damage.

Serumic calculus is dark and extremely hard, and deposited in minute scales or nodules.

(*c*) *The gingivitis and destruction of peridental membrane.*—The inflammation of the gums either produced directly by the irritation of the deposits of tartar, or which having preceded it is aggravated by it, is familiar to us all—the tumid, reddened, spongy gums, bleeding at the slightest touch, and on cleaning the teeth. Up to this point there has not of necessity been any pyorrhœa. The gums do not exude pus when pressed. But further changes soon take place. The first effect of inflammation of

the gums, as of other tissues, is the accumulation of leucocytes and the proliferation of the connective-tissue elements. The effect of this state of things is mechanically, by mere pressure and by blocking of vessels, to cut off the portion of the blood supply of the peridental membrane which is derived from the gums. At the same time the morbid process extends to the peridental membrane, following the course of a blood-vessel or a lymph path, and this structure also becomes swollen by proliferation of leucocytes and connective-tissue corpuscles. As it has been, as we have seen, partially deprived of its vitality and blood-supply by the gingivitis, the effect of the pressure to which it is subjected by its increased bulk between the two unyielding surfaces, tooth and alveolus, is to cause local necrosis. The immediate result is the liquefaction and breaking down of the diseased areas, and the formation of a cavity or pocket between the alveolus and the root of the tooth. This terminates this stage.

(*d*) *The streptococcus invasion.*—Up to this point, as we have remarked before, there has been no

production of pus. But this condition of things does not last long after the pockets have been once formed. There is soon infection of their walls by the pus-forming organisms always present in the oral cavity, and true pyorrhœa is established. At this stage a careful examination with a thin flat blade will show that destruction of the peridental membrane is taking place, as the instrument will pass up further than it should along the side of the tooth, into a little pocket which has already formed, and contains pus. These pockets are the characteristic feature of the disease; they follow the length of the fibres of the peridental membrane, and run up alongside the roots of the teeth. They may be single or multiple, and may be met with on the lingual, buccal, or proximal sides of the teeth. It not unfrequently happens that the entire apex of a root is denuded, whilst the tooth is held in its place by a strip of membrane on the other side, which has as yet undergone little change. The tendency eventually is to the destruction of the whole peridental membrane, as the pockets slowly widen until they encircle the

roots. The ultimate result being total loss of the teeth.

(*e*) *The absorption of the bony tissues.*—At the same time that the changes which we have described are taking place in the peridental membrane, absorption of the alveolar wall is going on. In some cases, whilst the inner portion of the alveolar wall is being destroyed, deposit of new bone is taking place on its outer surface. The result of this is that the alveolar wall becomes lipped, and by its everted edge holds the gum away from the tooth. The destruction of bone appears to be a process of true absorption rather than a molecular necrosis, and is the result of an absorptive action of the membrane itself, possibly through the agency of osteoclasts. This is due to the irritation produced by the products of inflammation.

The atrophy of the pericemental membrane keeps pace with the absorption of the alveolar process, so that a V-shaped orifice is found on examination, the floor of which is the exposed root of the tooth. If the tooth is extracted early in the disease there will

not be found as a rule any nodules of tartar on the root; but if it is examined towards the close of the disease, when the whole of the root is exposed, small nodules will be found scattered over its surface. These are quite distinct from the serumic calculus which forms under the edges of the gums, and correspond to individual necrotic areas in the pericementum, in each of which is a deposit of lime salts.

From Gouty Diseases in the Substances of the Peridental Membrane.

In such a case the affection commences as gouty disease of a part of the pericemental membrane. A vessel becomes obliterated, and necrosis of the small area supplied by it takes place. At this stage there will probably be no symptoms except, perhaps, pain in the alveolar process adjacent to the tooth. An examination of the gums will reveal nothing abnormal. There is no gingivitis, and there is no breach of continuity of the peridental ligament. The next stage will be a painful swelling over the root of the tooth.

The patient is said to have a gumboil, and there are all the symptoms of acute apical pericementitis. The swelling is either opened with a bistoury, or breaks of its own accord, and a thick glairy fluid escapes. Sometimes the discharge consists of pus, but this is rare except there is also an exposed pulp, through which the pyogenic organisms can get access. On examination with a probe, we shall find that there is a loss of the underlying alveolar wall, and that we can feel at the bottom of the cavity the root of the tooth denuded of its pericementum. Upon this root we shall often find a calculus. This, as was demonstated by Pierce, will, in the great majority of cases, contain a large proportion of urates. If not properly treated, the disease will progress until the pericemental membrane is perforated towards the gum margin, and a pocket is formed.

In other cases the course of the affection is different. Instead of perforation of the alveolar wall, we shall have extension of the gouty necrotic process towards the margin of the peridental membrane, which becomes perforated, and a pocket is formed

which undergoes infection with pus-producing organisms.

Another way in which pyorrhœa may arise is from death of the pulp of the tooth. The first event in pyorrhœa from this cause is death of the pulp of the tooth. By infection through this dead pulp we next get inflammation of a limited portion of the peridental membrane near the apex of the tooth. Absorption of the alveolus immediately over it will then take place, and we shall get a painful swelling on the outside of the gum, which will either eventually break or be opened by the surgeon. In either case it will be found that the root of the tooth is exposed, and upon this will usually be found some nodules of tartar. It has been held by some writers that such tartar is really an attempt by nature to repair the damage done by the deposition of lime salts; but, whether such is the case or not, it will be necessary to remove them before a cure can be effected.

The subsequent course of an affection of this kind will resemble other cases of pyorrhœa if it be not

arrested by appropriate treatment. The morbid process will extend towards the free edge of the peridental membrane, and a pocket will eventually be formed.

The Importance of the Study of Pyorrhœa Alveolaris to the Practitioner of Medicine.

Athough, strictly speaking, this condition lies within the province of the dental surgeon, yet it should be of great interest to the physician, since upon it may depend many disturbances of the whole system, and particularly of the digestive organs. So much is this the case, that I venture to assert without fear of contradiction, that the presence of a pyorrhœa may often afford a valuable clue to the elucidation of departures from health which have baffled our powers of investigation.

Pyorrhœa alveolaris may act in three different ways in the causation of disease.

1. The pus with its multitude of putrefactive

organisms and decayed food remnants from the pus pockets may be swallowed and—

(*a*) Act locally upon the stomach wall.

(*b*) Set up fermentation of the stomach contents.

2. The toxines generated in the mouth may be absorbed directly into the system through the mucous membrane of the mouth or stomach.

3. The presence of the local condition in the mouth may favour the growth and development there of pathogenic organisms, and thus render the patient more liable to contract certain infectious disorders, notably influenza.

Before studying these effects in detail it will be as well to briefly pass in review what is now known as to the micro-organisms which inhabit the mouth.

That these micro-organisms are very numerous we should naturally expect when the following facts are taken into consideration :—The mouth is constantly in contact with the atmosphere ; it offers the necessary conditions for the cultivation of disease germs, viz. warmth, moisture, and an alkaline reaction ; and

there is usually present in the mouth plenty of culture material upon which they can grow, in the form of food débris, &c.

Such being the case, it is not surprising that we have the following list of micro-organisms which have been found in the mouth, and specially named by those who have observed them :

Bacillus buccalis maximus, Bacillus α and β (Galippe), Bacillus prodigiosus, Bacillus violaceus, Bacterium cerasinum, Bacterium coli commune, Bacterium gingivæ pyogenes, Bacterium termo, Diplococcus Hauserii, Iodococcus vaginatus, Leptothrix buccalis, Leptothrix innominatus, Micrococcus gingivæ pyogenes, Micrococcus ochraceus, Micrococcus Reesii (Rosenthal), Pneumococcus of Friedländer, Pseudo-diphtheritic bacillus of Roux and Yersin, Proteus vulgaris, Saccharomyces cerevisiæ, Sarcina aurantiaca, Sarcina lutea, Sarcina viridis flavescens (Rosenthal), Spirillum sputigenum, Spirochœte dentium, Staphylococcus albus, Staphylococcus aureus, Staphylococcus citreus, Streptococcus, Vibrio rugula, and Vignal's bacillus.

In addition to the above lengthy list there are many other varieties present in the mouth which have not yet been sufficiently studied to enable names to be given to them. Although many of the micro-organisms of the mouth are merely temporary visitors, yet others are met with so constantly that they must be looked upon as permanent inhabitants.

According to Miller,* in the healthy mouth we meet with chiefly the Leptothrix innominatus, Bacillus buccalis maximus, Iodococcus vaginatus, Spirillum sputigenum, and the Spirochœte dentium; whilst in cases of pyorrhœa we find in addition the Micrococcus and Bacterium gingivæ pyogenes. We shall also find sarcinæ and streptococci.

Notwithstanding the number of organisms in the mouth, it is a curious fact that it is usually not very septic. And our knowledge of the reasons for this we owe to the important researches of Dr. Hugen-

* Miller, 'Inaug. Diss.,' Berlin, 1887; 'Centralbl. für Bacteriologie und Parasitenkunde,' i, 47, 87; 'Archiv für exp. Pathol.,' xvi, 291, 1882; 'Deutsch. med. Wochenschr.,' x. 395, 1884; 'Die Micro-organismen der Mundhöle,' Leipzig, 1889.

schmidt* which were made in Professor Metchnikoff's laboratory at the Pasteur Institute. It was at one time supposed that the saliva possessed a bactericidal action, and it was by this means that the growth of organisms in the mouth was kept within bounds. But that this was the case only to such a very slight extent as to be practically ineffective for keeping the mouth aseptic is proved by the experiments of Sanarelli. Curiously enough these demonstrate exactly the opposite of what they were intended to do by their author. He published them to show that saliva *had* the power of destroying micro-organisms. He found that saliva carefully filtered through Pasteur's porcelain candles, so as to entirely free it from germs of all kinds, had the power of killing certain pathogenic organisms, notably the Staphylococcus pyogenes aureus. But this occurred only with such small quantities that its action upon the germs of the mouth, which according to Miller frequently amount to 140,000,000 in an ordinary unclean mouth, may probably be neglected.

* Hugenschmidt, 'Dental Cosmos,' 1896, p. 797.

The explanation given by Hugenschmidt of the fact that the mouth is not more septic, is found in the reciprocal action of the bacteria and their secretions upon each other. Just as in the larger creation, nature provides against the undue multiplication of any special animal or insect, so in the microscopic world similar agencies are in operation which effectually limit by mutual destruction the inhabitants of the locality. Moreover, actual invasion of the tissues of the buccal cavity is prevented in the healthy mouth by the activity of the phagocytes in the mucous membrane, and by the property possessed by stratified pavement epithelium of continuously shedding and renewing its superficial layers.

Having now a clear appreciation of the micro-organisms of the mouth, we can proceed to study their evil effects upon the system. In doing so we shall follow the scheme already given.

1. *Pus, micro-organisms, and decayed food from the pus pockets may be swallowed.*

It has been thought for a long time that the

gastric juice had the power of destroying pathogenic organisms which are introduced into it; so much so, that Bunge enunciated the theory that the chief object of the gastric juice was the sterilisation of the stomach. But this is far from being the case, as not only theoretically can there be but a very short time during the digestive period when the gastric juice will contain a sufficient degree of free HCl to destroy these germs, but practically it is shown that many do actually escape destruction, since the mouth bacteria constitute, according to Lucksdorff, at least 3 per cent. of those found in the intestinal contents. This noxious material thus swallowed may—

(*a*) Act locally upon the stomach wall.

It is extremely probable, although it has not been absolutely proved, that some obscure cases of acute gastritis may be set up in this way. Hemmeter * has the following:

"The direct causes of the rarer idiopathic phlegmonous gastrites are unknown, ... but judging from anatomical specimens, are probably

* Hemmeter, 'Diseases of the Stomach,' Philadelphia, 1887, p. 410.

bacterial invasions of the submucosa, principally from pyogenic cocci, which find portals of entry through lesions in the superficial epithelium of the stomach, such as occur in most gastric diseases. . . ."

What more likely, then, that under certain conditions, when the other factors are present, we may have an acute gastritis set up directly by the material from decayed teeth and pus pockets which has been swallowed by the patient—the conditions necessary being such a low degree of acidity in the gastric juice that organisms escape destruction, and the presence of some slight local lesion through which they can gain admittance to the submucosa.

As regards chronic gastritis, I think that we take it as proved that it is often caused by swallowed micro-organisms and pus. In these cases it is apparently not the micro-organisms themselves, but their irritating products which set up the gastric irritation. This is especially true of the yeasts, which produce excessively irritating products. But these products of fermentation come under the heading of—

(*b*) Fermentation of stomach contents produced by micro-organisms swallowed.

The most important of these are the fermentations produced by the different species of yeast plants, the lactic acid and the butyric acid fermentations. And, since we have seen that the lactic acid micro-organism is invariably met with in connection with tartar and pus pockets, it is easy to understand how useless it is to attempt to cure a chronic gastritis while the mouth is in an unhealthy condition. In these cases the HCl of the gastric juice is usually in defect, and consequently the antiseptic power of it practically *nil*. What good, then, can we expect to do with the small doses of antiseptic drugs which we introduce by the mouth, if we at the same time allow the patient to swallow fresh germs with every meal? In such cases I have seen the happiest results follow the use of a weak solution of tincture of iodine immediately before each meal as a mouth-wash.

The power of pyorrhœa alveolaris to produce aggravation of existing gastric trouble reaches its maximum in cases where there is habitual retention

of food residues. This happens when the muscular walls of the stomach are in a state of atony, and also when there is some pyloric obstruction which prevents the organ emptying itself. In both these conditions there is eventually produced a dilatation of the stomach, with the result that it is never completely emptied. In the management of this condition one of the chief problems which confront the physician is to limit the fermentative processes upon which so much of the discomfort of the patient depends. With appropriate diet, lavage, and antiseptics, much can be accomplished; but it is easy to see how much more difficult the task becomes if the patient is continually swallowing bacteria and pus cocci with his food. I am firmly convinced that enough attention has not been given by the practitioner to this aspect of the subject, and I am afraid that numbers of cases of atony and dilatation of the stomach are treated in every-day practice without that careful examination of the mouth which should precede all attempts at systematic treatment by the methods enumerated.

It is not improbable that pyorrhœa alveolaris may be a factor in the perforation of a gastric ulcer. Hemmeter * has the following:

"In the gastric ulcer, however, there is another kind of bacterial infection, which is not accompanied with the signs of active inflammation, and is termed by some authors bacterial necrosis. The process is characterised by the invasion of bacteria, usually in the lower depths of the mucous membrane, by their growth and subsequent necrosis of the tissue. Although the secretion of HCl is germicidal to many bacteria, it must be remembered that their spores are not destroyed by it, and that the invasion may take place during the period of rest of the glands in the intervals of digestion, when no or very little HCl is secreted. There is room for the suggestion that the primary necrosis is due to bacteria, and the ensuing ulceration caused by the action of the gastric juice."

A subject which requires working out is the possible relation between pyorrhœa and empyema of

* Hemmeter, op. cit., p. 469.

the antrum of Highmore. It is not inconceivable that this affection may sometimes be directly initiated by it. We have seen how one form of pyorrhœa commences by death of the pulp, and how in some way the tissues outside the apex of the tooth become infected. If it should happen that the affected tooth was one which projected into the cavity of the antrum, as is sometimes the case, we might readily get an empyema set up.

Again, it used to be a common practice—and is still in favour with many surgeons—to extract a tooth and perforate into the antrum in cases where there was reason to believe that there was a collection of fluid in that cavity. If that tooth were the subject of pyorrhœa it would be almost impossible to prevent infection of the antrum by bacteria carried up by the instrument, however thoroughly it had been sterilised before the operation.

A lengthened experience has convinced me that a very large proportion of the indigestions met with among the poorer classes such as throng the out-patient rooms of hospitals may be explained by the

very common occurrence of pyorrhœa alveolaris in these cases. Such patients very commonly neglect their teeth until they experience pain, and many cases of the severest grades of pyorrhœa may be met with in one afternoon's work at any of our large public institutions. What a farce it must be, then, to treat these unfortunate patients, as I have often seen done, by the routine administration of Mistura Gentian. Alk. or Mistura Bismuthi Sed., without even a cursory glance at the mouth to ascertain the condition of the teeth !

There is another way in which pyorrhœa alveolaris may injuriously affect the system, and that is—

2. *By the direct absorption of pus into the system.*

To what extent this occurs is at present doubtful, Some authorities, notably Herschell, believe that gastric neurasthenia may be thus explained. He says : * "There is good reason to believe that many of the chronic indigestions which come under our

* Herschell, 'Indigestion: an Introduction to the Study of the Diseases of the Stomach,' second edition, p. 54, 1895. London: Baillière, Tindall and Cox.

notice are due to the continual absorption of pus into the system either from a pyorrhœa alveolaris or from an abraded cervix uteri. . . . The subject is at present undecided, and offers a valuable field for further investigation. In such cases we should expect to find some of the other signs of the absorption of toxines, such as pigment spots on the arms, tachycardia, goitre, neuritis of the sixth left intercostal in women, and peptonuria. Urticaria or some other skin eruption may also be present."

Sufficient attention has not been given to the probability that—

3. *A pyorrhœa in the mouth may favour the growth and development of certain pathogenic organisms, and thus render the patient more liable to contract certain infectious disorders.*

It is quite certain that bacteria vary much in virulence at different times, and under conditions which at present have not been worked out. For example, the Bacterium coli commune, which we have shown to be a constant inhabitant of the oral cavity, is apparently usually harmless; but under

certain circumstances, such as possibly a pyorrhœa or inflammatory condition of the gums, it may acquire virulence, and produce serious disturbances of the organism, such as colitis, dysentery, and cholera nostras. As regards Asiatic cholera, it has been pointed out by Metschnikoff that the specific organism will multiply much more rapidly in the intestine if at the same time there is a larger amount than normal of yeasts and sarcinæ. We may take it, then, that the presence in the mouth of abnormal quantities of these fungi should theoretically increase materially the chance of the individual contracting this disease. The lesson pointed out is obvious. In presence of an epidemic it will be a matter of common precaution to have the mouth set right, and any pyorrhœa cured.

A subject which, however, strikes nearer home is epidemic influenza, which apparently we shall have ever with us. It is now almost proved that the specific germs of this affection gain an entrance into the system through the mouth. And it is a subject worthy of the fullest investigation to ascertain what

effect, if any, a foul condition of the mouth may have in affording them the necessary conditions for their growth. In the meanwhile we shall be on the safe side if we insist on all our patients putting their mouths in order.

Diagnosis and Treatment.

Having now a theoretical acquaintance with pyorrhœa, and its effects, both locally and upon the system generally, the next thing is to learn to recognise it when it comes under our observation. But since, as in other affections which are met with in daily practice, a scientific diagnosis comprises, not only the recognition of the local condition, but also the estimation of any underlying dyscrasia, so, we must not content ourselves with a simple diagnosis of pyorrhœa. We must ascertain whether it is merely a local condition which can be removed by local treatment, or a local expression of a constitutional vice. We must therefore study in succession and separately the local condition and the consti-

tutional state of the patient. We shall take these in order.

The Local Condition.

To cure pyorrhœa it is important to recognise it in its earliest stage. Unfortunately, this is rarely done by the dentist, who overlooks the affection until streptococcus invasion has taken place and pus pockets have formed. The most favourable time for cure has then passed. In order to simplify the study of pyorrhœa we shall divide it into four types, and discuss the diagnosis and treatment of each *seriatim*. The treatment of the first three of these can quite well be carried out by a medical man with no special dental training or knowledge.

Type 1.—This is the earliest stage of pyorrhœa, and there is not of necessity any visible tartar on the teeth. There is merely a gingivitis present, which will, if not checked at this stage, inevitably proceed to ulterior change. The margins of the gums are inflamed, and beneath them are deposited, on the

necks of the teeth, dark and hard nodules or scales of the so-called serumic calculus. If not properly cured, inflammatory degeneration of the pericementum will inevitably ensue, with eventual loss of the teeth. The edges of the gums are red, swollen, and spongy, and bleed when roughly touched either with a tooth-brush or the fingers. The act of sucking exerted in smoking a pipe will also cause them to bleed readily. It may, or it may not be possible, to cause pus to exude from under them by pressing with the finger tips.

The affection in this early stage, when it is not strictly pyorrhœa, because pus is usually not present, is frequently met with in debilitated and anæmic subjects, and among those convalescing from acute diseases. The diagnosis is made certain by the finding of serumic tartar under the edges of the gums. This, as has been already remarked, is deposited from the serum poured out as discharged, and is a certain sign of inflammation.

The first thing, then, after having discovered the presence of a gingivitis, is to examine for serumic

tartar, and thus find out whether the affection in question is a trivial condition, or whether it is the first stage of pyorrhœa alveolaris. The instruments required for this purpose are a set of Cushing's scalers (see Fig. 4). Some of these instruments, it will be noticed, have terminal hooks, as shown in Fig. 4, *d, e, f.* The method of using them will be described presently.

The treatment of pyorrhœa in this stage is not difficult, and usually successful, and comprises the removal of the tartar, the application of an astringent to the inner margins of the gums, and the prescription of a mouth-wash for daily use.

The removal of the tartar.—To avoid repetition, the directions for doing this will be deferred until types 2 and 3 are discussed.

The astringent application.—My favourite for this condition, and one which I have used for many years, is sulphate of copper reduced to an extremely fine powder. To apply it properly you require a small point of wood, about half an inch in length, tapering to a fine extremity. Such a piece of apparatus may

be made from one of Bryant and May's large matches, whittling the end down to the proper shape, and finishing it off with a piece of fine sand-paper. It is conveniently set at right angles in a special holder made on purpose, which may be procured at any of the dental depôts. The following is a full-size photograph of the apparatus (Fig. 1) :

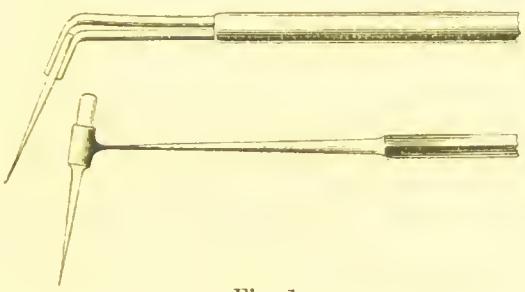


Fig. 1.

You will also require about two drachms of finely-powdered sulphate of copper in a small wide-mouth stoppered bottle. You must also have ready to your right hand a piece of blotting-paper, a small glass of cold water, and a large tumbler full of tepid water. Set the patient facing a good light, so that the inside of the mouth is well illuminated. Now, having selected the tooth on which you propose to operate,

dip the wooden point into the water in the small glass, remove surplus moisture by touching it with the blotting-paper, and dip it into the bottle of sulphate of copper, some of which will adhere to it. Now wipe off the powder on to the neck of the tooth close to the gum margin, and gently pack it down under the edges of the gum. Continue this process until you have treated all the affected teeth, and a blue line above the margin of the gums shows that the operation has been properly performed. Now let the patient rinse the mouth with warm water, which you have in readiness, and send him away with the following prescription :

℞	Glyc. acid. carbol.	...	...	ʒiv
	Glyc. acid. boric.	...	...	ʒiv
	Potassæ chloratis	...	...	ʒij
	Euthymol	...	...	ʒiv
	Aqua anisi ...	...	...	
	Aqua menth. pip.	...	āā	ad ℥viij

Mix.—To be used as a mouth-wash night and morning.

It will also be found to materially expedite the

cure if the patient use a few drops of listerine upon the tooth-brush. Indeed, a very good mouth-wash may be made by mixing half an ounce of this preparation with six ounces of water.

The packing with sulphate of copper should be done several days in succession, then every alternate day for a week, and then twice a week for another fortnight. The patient will then probably be cured.

Type 2.—On examining the patient's mouth, we see the sides of the teeth incrusted by well-defined masses of tartar, the lower edges of which are in contact with the margins of the gums. These are inflamed, reddened, and eroded. On close examination one may see between the edges of the gums and tartar respectively, a narrow zone of uncovered tooth bathed in pus. On rinsing away this pus with the stream of a syringe, we are unable to squeeze any more from under the edges of the gums. On passing an instrument under the edges of the gums we may, as in type 1, find nodules of serumic calculus, but on examining very gently under the edges of the

gums with a blunt instrument, as in type 1, we cannot pass it down along the side of the root. There are, therefore, no pockets, and the affection is an example of type 2, and not type 3. The pus comes from the ulcerated edges of the gums, and is the product of the inflammatory process. The pyorrhœa is only in the incipient stage, as the peridental ligament is intact, and we should readily cure it.

The local treatment will be exactly the same as described for type 1, with the addition of first removing the tartar from the sides of the teeth, and a preliminary treatment to subdue the local inflammation before the packing with sulphate of copper is commenced.

It will at this juncture be convenient to describe the proper method of removing tartar from the teeth. It is quite an easy operation if performed with a correct technique and with suitable instruments, and well within the power of any surgeon without any special knowledge of dentistry.

The necessary instruments are the following:

(*a*) Two or three crooked or hoe-shaped instruments of various sizes for the removal of the bulk of large concretion, by a pulling motion (Fig. 2).

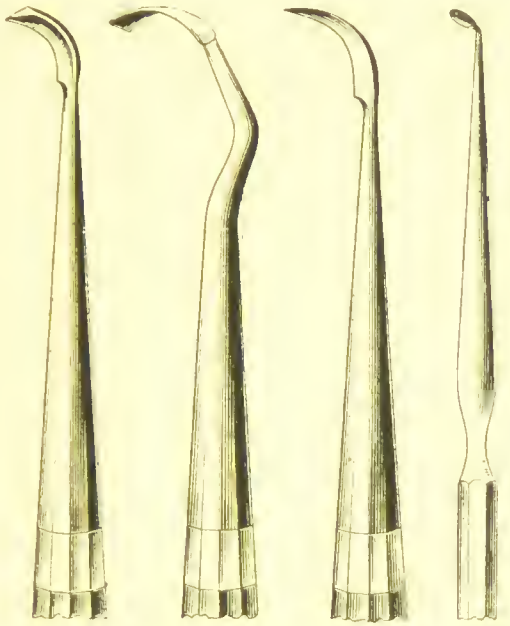


Fig. 2.

(*b*) A set of Howe's scalers (Fig. 3) to be used in a similar manner for removing smaller portions; and

Fig. 3.

(*c*) a set of Cushing's scalers for removing with a pushing and pulling motion the last particles of salivary and serumic calculi (Fig. 4). Some of these are also used in probing for pockets.

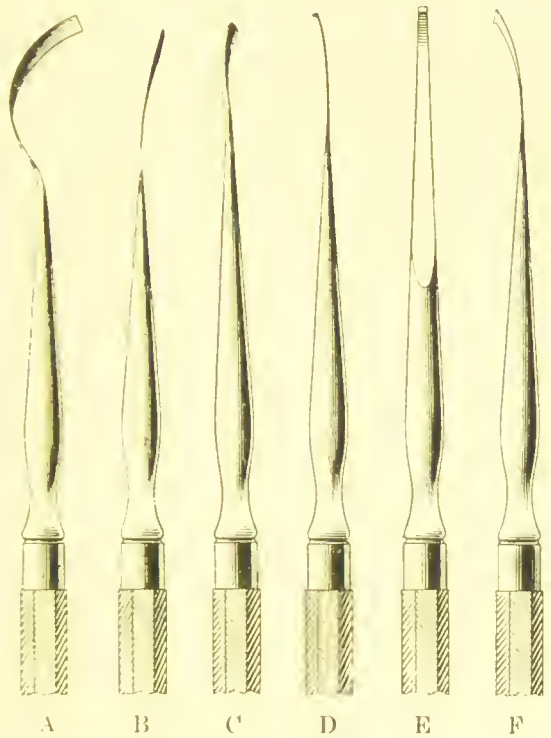
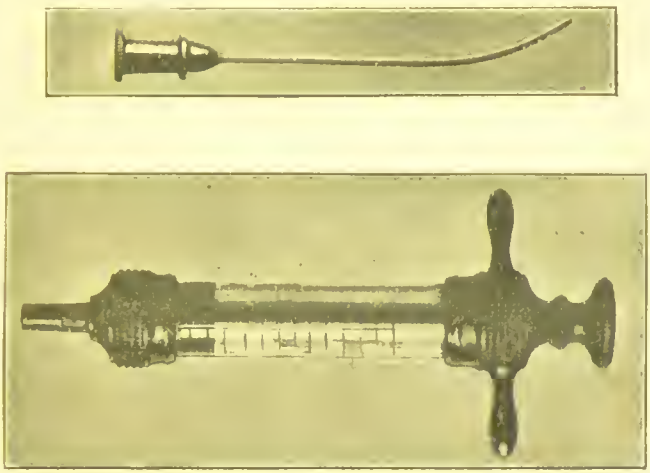


FIG. 4.—These scalers are of two kinds. Some, as A, B, C, are for detaching the calculus by pushing. Others, as D, E, F, are provided with a terminal hook, and are used for hooking out the *débris* detached by the former.

In the treatment of this stage of pyorrhœa we shall also require—

(*d*) A hypodermic syringe, provided with a special cannula (Fig. 5), the cannula to be made of gold, and very fine. This is used for the application of medicated solutions to the peridental membrane, and to the pockets met with in types 3 and 4.



Fig, 5.

There is a right and a wrong way of removing tartar. In removing the large salivary calculus, a hoe-shaped instrument must be taken, and the end passed under the mass of tartar where it touches the

gum. The instrument is then pulled towards the crown of the tooth. The accompanying illustration will make this plain (Fig. 6).

Salivary tartar around the sides of the teeth can readily be removed by the novice, but the dislodg-

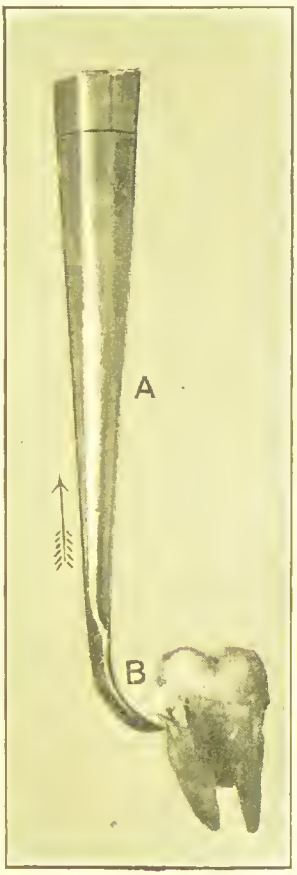


Fig. 6.—A Scaler. B Calculus, † Direction of pressure.

ment of the small hard scales of serumic calculus under the edges of the gums, and in pockets, is much more difficult. The technique is as follows (see Fig. 7) :—Take a Cushing scaler, and holding it

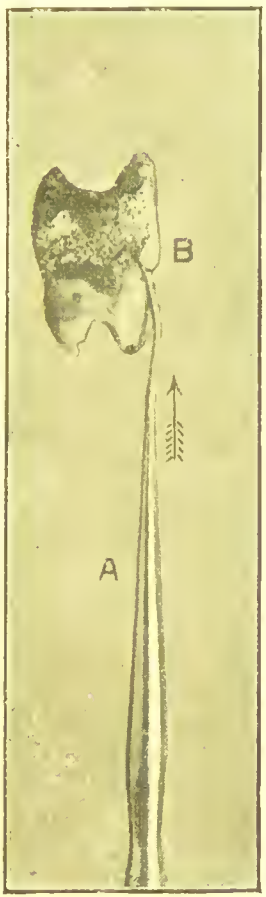


Fig. 7.—A Scaler. B Calculus. † Direction of pressure.

between the thumb and forefinger, with the concavity of the blade towards, and its long axis corresponding with that of the tooth, pass it gently down the sides of the latter, exercising a gentle pressure against it. When its extremity reaches a scale of tartar it will be arrested. The chisel edge of the scaler is now in contact with the point where the nodule of tartar joins the tooth. If you now continue the forward movement of the scaler, the calculus will start right away from the tooth without any particular force having been used. Perfect control must be kept over the instrument in order to prevent it from suddenly moving forwards as soon as the resistance offered by the scale of tartar has been removed, and thus wounding the peridental membrane. The process is the same when pockets are present (see type 4). The instrument is carried right along the root in the axis of the tooth, and every particle of tartar is loosened as it is encountered. Then, with a Cushing hooked scaler of suitable size and shape (see Fig. 4, *d, e, f*), remove the loosened and detached scales with a drawing or pulling movement.

It is most important that *every particle* of tartar should be thoroughly removed, and a careful search must be made for it, as any small particle overlooked will certainly keep up the irritation, and effectually prevent the healing process.

After the removal of the deposits, peroxide of hydrogen solution should be syringed under the edges of the gums, as by its effervescence it will wash up the particles which have been detached, and materially assist their removal. The gums may now be packed with sulphate of copper as already described, or may be painted with tincture of iodine every other day for a week.

In removing tartar at this stage of the disease, it is not necessary to avoid scarifying the gums; in fact, this will do good by removing blood, but one must be *very careful* not to wound the peridental membrane, as this is *intact;* and if you should be so unfortunate as to puncture it, you will open the door for bacterial invasion, and possibly produce type 3.

Type 3.—In this stage of the disease the peridental membrane has had its continuity destroyed

at certain points, and consequently pockets have formed which have been infected with pus-producing organisms. It differs from type 4 insomuch that there are, however, no loose teeth.

The gums may possibly be merely reddened at the margin, but when pressure is applied to them pus will be seen to well up along the sides of the teeth.

A careful examination with a thin flat blade will show that destruction of the peridental membrane has taken place, as the instrument will pass further than it should along the sides of the tooth into little pockets which have formed and contain pus. These pockets are characteristic of this stage of the disease. They follow the length of the fibres of the peridental membrane, and run up alongside the root of the tooth. They may be single or multiple, and may attack the lingual, buccal, or proximal sides of the teeth. The tendency is eventually to the destruction of the whole peridental membrane, as the pockets slowly widen, usually irregularly, until they encircle the teeth. At the same time that these changes are going on in the membrane, gradual

absorption of the edges of the alveolar wall is taking place. In some cases, whilst the inner portion of this is being destroyed, deposit of new bone is taking place on its outer surface. The result of this is that the alveolar wall becomes lipped, and by its everted edge holds away the gum from the tooth. On examination with the instrument then, we shall—

(*a*) Find pockets.

(*b*) Establish the presence of nodules and scales of tartar on the root of the tooth in the pocket.

(*c*) In many cases be able to make out the eroded and roughened edges of the alveolus.

As regards treatment, the first thing to do is to remove all deposits of tartar in the manner already described. There are, however, several points which must be kept in mind, if we would make an unqualified success.

1. Commence at one tooth, and remove every particle of tartar before proceeding to the next.

(2) Whilst doing this, syringe every now and again with peroxide of hydrogen, of which the best form is

pyrozone. The effervescence will not only mechanically bring to the surface the *débris* which you have removed, but will show the position of the pus pockets. Peroxide effervesces with pus.

3. In cases where it is specially difficult to remove all the particles of tartar, we may derive great assistance from the use of a 1 in 20 solution of sulphuric acid syringed into the pockets. Allow this to remain in a few minutes, and then neutralise with a solution of carbonate of soda introduced in the same manner.

4. It is a good practice, as a preliminary, to pack the pockets with cotton wool, wetted with a 3 per cent. solution of pyrozone containing a little cocaine, as this both dilates and blanches the pockets and renders the subsequent operation comparatively painless.

Having now removed all deposits of tartar, and scraped the edges of the alveoli (if we have found it necessary), we have several alternative methods of treating the pockets to secure healing, and the adhesion of the gum to the teeth. There are a

good many remedies, and unfortunately too many people are trying to make reputations by proposing new ones. The following have stood the test of practical experience, and are indicated under various conditions.

(*a*) The pockets may be simply syringed out twice a week with a strong solution of peroxide of hydrogen (pyrozone). The patient in the meantime uses a 3 per cent. solution as a mouth-wash.

(*b*) The pockets may be syringed out with a solution of peroxide of hydrogen containing 1 gr. of perchloride of mercury to the ounce. Express this, dry the pocket with hot air, and then introduce a 30 per cent. solution of chloride of zinc. Repeat these two applications every day for a week. You now direct the patient to attend every four days, and on each occasion syringe the pockets with peroxide as before; but, instead of the chloride of zinc, introduce with a brush a few drops of the following mixture :

R Ol. cinnamomi ... ... ℥iv
Ol. gaultheriæ ... ... ℥iv
Acid carbolic (crystals) ... ... ℥j
Mix.

At the same time you give the patient a prescription for this same mixture, but diluted with an equal quantity of oil of lemons or parolein, to be applied to the gums daily with a brush, and order him to use as a mouth-wash simple cinnamon water.

(*c*) The application of lactic acid is especially advocated by Younger, who claims to cure by this means twenty-four out of twenty-five cases ('Dental Cosmos,' 1896, p. 676)! The acid is warmed slightly, and injected with a platinum-pointed syringe clear to the bottom of the pocket. It is not washed out, but allowed to remain there. The gum is protected during the application by a piece of cotton wool, smeared with vaseline. He affirms that one treatment for each pocket is usually sufficient.

(*d*) Another method is to introduce pure sulphuric acid into the pockets with a platinum wire. Only the smallest quantity must be taken on to the wire,

and it must be carried by one movement right down to the bottom of each pocket. The pocket must be syringed immediately afterwards with a solution of bicarbonate of soda.

(*e*) Trichloracetic acid, in a 20 per cent. solution, has been strongly advocated by some writers, who claim that it is especially indicated when the edges of the gums are tumified and everted.

(*f*) Sudduth recommends packing the pockets with the following mixture :

℞	Zinci sozoiodat.	... ...	gr. viij
	Morph. acetat.	... ...	gr. ij
	Lanoline. ...	... ...	ʒj

Mix.

(*g*) A composition in the form of a syrupy brown liquid was devised by Dr. Berlioz, and communicated to the French Academy of Medicine in 1893. It is applied to the pus pockets with a camel-hair brush, and very good results have been recorded from its use. He applied the name to it of "steresol," and it has the following composition :

℞ Purified gum lac	270·0	grm.
Purified gum benzoin ...	10·0	,,
Balsam of Tolu	10·0	,,
Carbolic acid crystals ...	100·0	,,
Chinese oil of cinnamon .	6·0	,,
Saccharin...	6·0	,,

Alcohol to make 1 litre. Mix.

(*h*) The pus pocket may be regarded as a fistula, and treated with the application of the galvano-cautery. This very severe method of treatment would be of undoubted efficacy in cases which have resisted all other methods; but personally I have never needed it, as I have been quite satisfied with the results obtained by milder measures.

(*i*) Another method of treating this stage of pyorrhœa is by means of cataphoresis. There can, I think, be no doubt that whatever applications give good results, when applied in the ordinary manner, will produce much better ones when applied cataphorically. The difficulty in treating pus pockets is to cause the medicament used to penetrate deeply

enough into the affected tissues to destroy all microbes. To do this in the ordinary way we are forced to use strong acids and caustic applications. By cataphoresis we can drive, by means of the electric current, a mild antiseptic into the gum, and at once destroy all germs, and stimulate the diseased tissues to a healthy action. The method of application is not difficult. The first thing is to apply a rubber dam or napkin to the mouth; the next is to dry the pus pockets and root canals with alcohol; cotton wool is then moistened with the fluid which we wish to apply, and packed round the teeth and into the pockets. A suitable platinum electrode is then placed upon the cotton, and the negative one upon some indifferent part of the body. A current is then passed for a few minutes of such a strength as will be just perceptible to the patient. There is no pain in this application and very little discomfort. (Full directions for this method of treatment are given in the 'Dental Cosmos,' vol. xxxix, in different articles by various authorities.) Many drugs may with advantage be used by cataphoresis. Personally, I use

chiefly oil of cassia, watery solution of iodine, euthymol and iodine, and euthymol iodine mixed with sanitas oil.

Whilst the surgeon is treating the pockets during the patient's visits, the latter should sedulously carry out an auxiliary treatment at home which may be comprised in—

(1) *Mouth-baths.—It is not sufficient*, and this point should be strongly impressed upon the patient *simply to rinse the mouth out with the solution used.* This should be retained at least five minutes in the mouth. In severe cases such a mouth-bath may be taken after each meal; later on, night and morning. The following are eligible formulæ:

(*a*) Solution of carbolic acid 1 in 100.

(*b*) Solution of chloral hydrate 1 in 100.

(*c*) One of Dr. Seiller's tabloids (Parke, Davis and Co.), dissolved in 2 oz. of water. These admirable preparations contain in varying proportions the bicarbonate, chloride, benzoate, and salicylate of sodium, with the oils of thyme, menthol, eucalyptus, and gaultheria.

(2) Daily massage of the gums with the fingers.

(3) The use of a suitable tooth-powder.

In all cases of pyorrhœa it is of the utmost importance to prescribe a tooth-powder for habitual use by the patient, which is especially adapted to the conditions obtaining in the mouth. If this be acid, which should be ascertained by litmus paper, an alkali should predominate in the powder.

Either of the following are good :

℞	Cretæ precip.	℥iv
	Sodæ bicarb.	℥ss
	Pulv. radicis iridis	℥ss
	Pulv. saponis alb.	℥j
	Acidi carbolici	♏xxx
	Ol. eucalypt.	♏xxx
	Ol. caryophylli	♏xx
	Ol. limonis	♏x
	Ol. menth. pip.	♏xx
	Ott. rosarum	♏vij

Mix.

R	Saccharin	0·20 grm.
	Thymol	0·1 „
	Acid. boric.	2·0 „
	Salol	4·0 „
	Pulv. saponis alb.	10·0 „
	Calc. carb., magnes. carb.	āā 20·0 „
	Ol. menth. pip.	0·1 „

Mix.

Type 4.—In this group we find the same conditions as in the preceding type, but with the complication of loose teeth. The affected teeth project further out of the jaw than the sound ones, and appear to be forced partially out of their sockets. Such is really the case. The growth of inflammatory material which subsequently becomes converted into bone, gradually diminishes the depth of the sockets, with the result that the teeth are gradually extruded, being often partially rotated and distorted in the process.

The treatment of this form or rather stage of the disease can only be efficiently treated by an expert

in dental surgery; but as the results obtained are little short of miraculous, it will be of interest to mention the means employed.

The treatment consists in extracting the affected teeth, opening up the pulps, and removing all pulp and nerve tissue from the pulp cavity and root canals, treating and filling these with an appropriate antiseptic material, filling the crown with a suitable stopping, sterilising the tooth, drilling out the socket with a proper instrument, replacing the tooth, and retaining it *in situ* by a specially prepared splint, until bony union has taken place between it and the alveolus.

Of course this procedure has been preceded by the treatment of the other affected teeth as in type 3. In cases where teeth are merely loose and do not project to any great extent, good results may be obtained by fixing them in position either by simply tying together with silk, or by the application of a proper splint.

Irregularities in the bite should be corrected. If any tooth strikes too hard, it is a good plan to put

temporary caps on the adjacent ones on each side to relieve the pressure.

This replantation of teeth has been known to the dental surgeon for a very long time, and we really do not know who originated it. It is not used as much as it deserves to be, as it requires that the dentist should have considerable surgical skill and knowledge. The important point to bear in mind in order to make a success is that everything must be absolutely aseptic. One of the best solutions to use is hydronapthol, 1 in 300, although chinosol will probably replace it. The operation should be performed by the following steps :

1. Place the tooth in the antiseptic solution, and raise to 180° Fahr.

2. Take the tooth out of the fluid and wrap it in floss silk, to protect it during the further manipulations.

3. Remove the pulp, and fill the tooth in the usual manner.

4. Drill out the new socket for the tooth.

5. Place the tooth in position.

6. Take a plaster impression and make a metal splint to go over both surfaces of the tooth.

In the front teeth it is a good plan to insert a wire into the pulp cavity, the end of which projects a quarter of an inch from the lingual surface of the tooth. The edge of the splint can then be passed between this and the tooth, and will hold it firmly in position.

The Treatment of the Constitutional State.

At the same time that local measures are being adopted to get the mouth into a healthy condition, we must employ constitutional treatment to remove any dyscrasia which may possibly cause the pyorrhœa. And it is here where the dentist often fails. Not having the technical knowledge to estimate the physical condition of the patient, he attempts repeatedly, persistently, but usually fruitlessly, to attack the disease by local measures. He scrapes away the accumulation of tartar, and when it re-

appears, as it shortly does,—*the cause being still there,*—he simply repeats the process.

It is necessary, therefore, to bear in mind that we must treat seriously any abnormal nutritive state or cachexia which may be present. As my readers are medical men, it will be quite unnecessary to do more than simply pass in review a few of the commonest of such conditions, and touch upon any point which can be elucidated from the standpoint of the dental surgeon.

The chief conditions which appear as factors in the production of pyorrhœa are :

1. *Affections or conditions which induce or perpetuate catarrh of the mouth or fauces.* By far the most frequent of these is chronic nasal obstruction, due to swelling or hypertrophy of the mucous membrane covering the inferior turbinated bones. As all specialists in diseases of the nose will tell you, this condition is more often than not unrecognised by the practitioner, owing to the fact that the said obstruction only occurs in many cases during the night. In the daytime there may be little or no actual stenosis,

and the surgeon on examining the nasal cavity with the speculum and frontal mirror, finds a fairly good passage. But at night, when the patient is asleep, the turbinals swell up, with the result that mouth breathing takes place until the patient awakes. The effect of this is to keep up a catarrhal condition of the pharynx, and secondarily of the oral cavity. The treatment, of course, I need not allude to.

(2) *Syphilis.*—In this affection it is possible for pyorrhœa to arise in two ways—from the cachexia produced in severe cases by the disease itself, and by the irritation attending the elimination of mercury and iodide of potassium from the gum margins. *It is, therefore, of the greatest importance to examine most thoroughly the mouth of the patient before commencing specific treatment.* Remove all tartar, treat all diseased roots, remove all roughnesses, and cure any pockets which may be present. It will be readily understood that the importance of this is not exaggerated when we bear in mind that we cannot carry on the specific treatment much beyond the point when gingivitis has appeared, without sali-

vating the patient. It thus unfortunately happens that when we commence a course of mercury without having first set the mouth in order, we have to stop it just when it is commencing to do good. If we start the course with the mouth in proper condition, we can delay materially the advent of the specific effects upon the gums by directing the patient to use several times during the day an astringent mouth-wash. The one in use at Aachen, during the cure there, is substantially as follows :

℞	Aluminis	ʒj
	Plumbi acetatis	ʒj

Dissolve the salts separately in 2 oz. of water, mix, and filter away the precipitated salts. To the filtrate add Aqua ʒij.

A favourite addition of mine to this is a small quantity of listerine or euthymol.

(3) *Gout.*—As we have seen, gout is probably a more frequent cause of true pyorrhœa than was until quite recently suspected. Such being the case, we should in all cases try to determine whether it is

present or not, as our treatment will certainly be modified by the conclusion at which we arrive. It would be out of place in this paper to discuss the signs of the gouty condition with which you are all familiar ; I will only say that no examination of a case of pyorrhœa should be deemed complete without a laboratory examination of the blood and urine. If any signs are obtained which will justify us in suspecting gout, the patient, at the same time that local treatment to the mouth is being carried out, should be placed upon an appropriate diet, and the hygienic measures and medicines prescribed which are indicated. I will only add that in my own practice, I have observed that daily baths, sometimes containing carbonate of soda, moist friction of the skin, plenty of exercise and fresh air, with limitation of alcohol, sugar, and fat, have materially hastened the cure of many of my cases of pyorrhœa.

INDEX.

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